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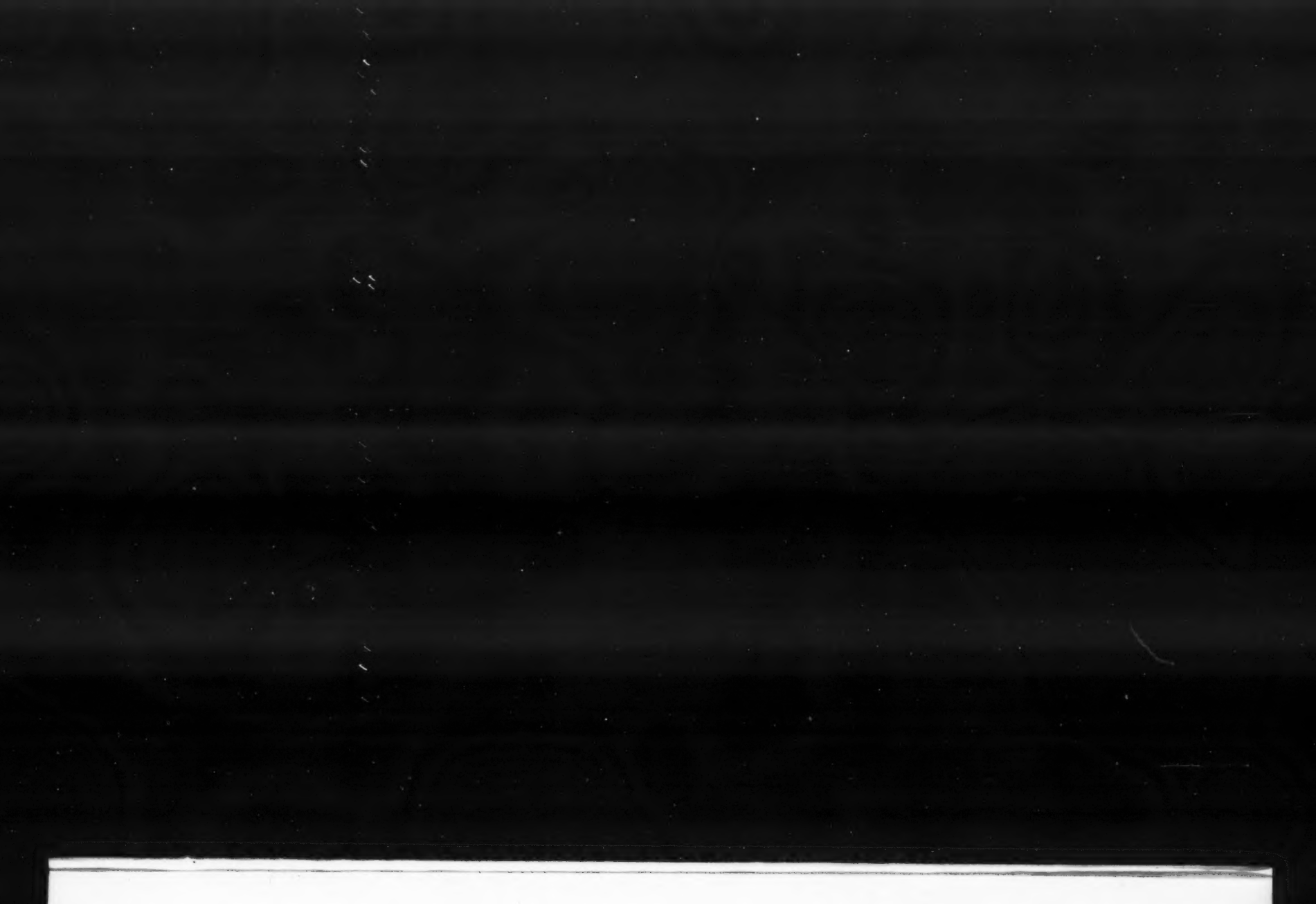

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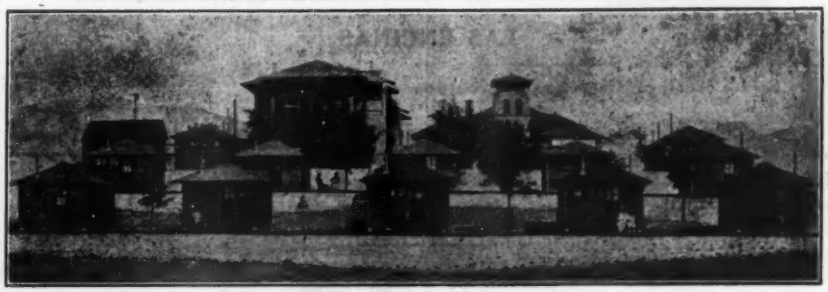

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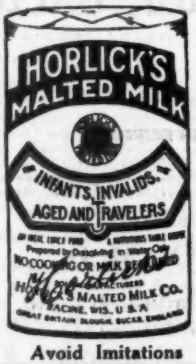

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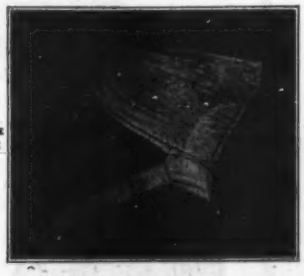

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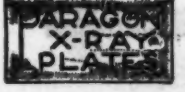

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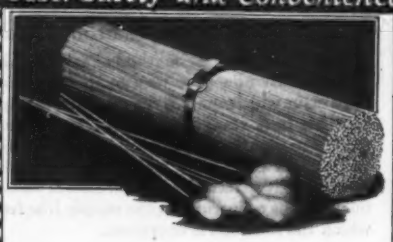

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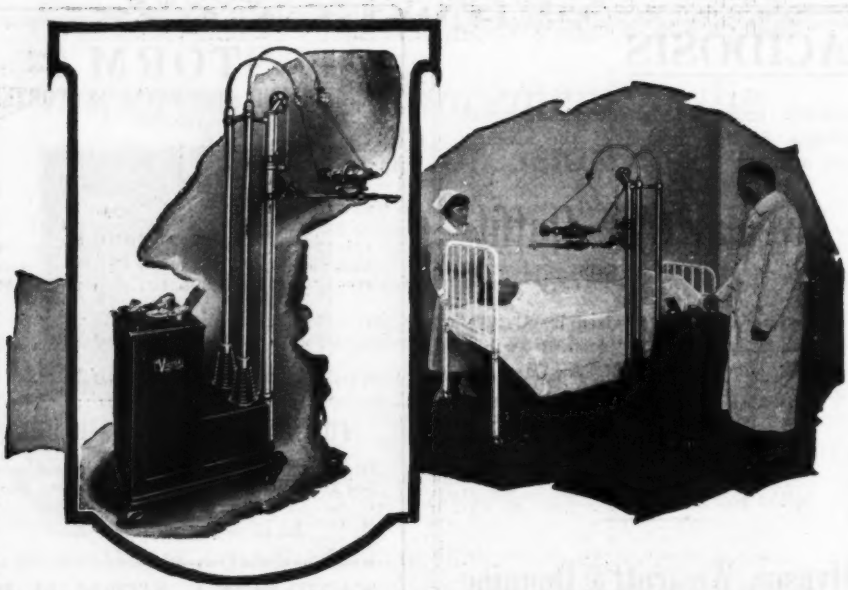

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IMPORTANCE OF DIAGNOSIS IN SOME LESIONS OF THE GENITO-URINARY APPARATUS*

CHARLES S. VIVIAN, M. D., Urologist, Southwest Clinic and W. WARNER WATKINS, M. D., F. A. C. P., Pathological Laboratory, Phoenix, Arizona.

So-called "silent lesions" of the genito-urinary tract occasion more mistaken diagnoses, and consequently, more unsatisfactory results, than any other diagnosable condition with which we have to deal. The adjective "silent" is a misnomer, however, as he who has his diagnostic ear attuned to the voice of the diseased kidney, ureter or bladder, will usually hear. As the human brain interprets the impressions which come to it over the auditory nerves, so must one's diagnostic judgment interpret the urological data furnished by the history, the urinalysis, the cystoscope and the x-ray. Urological literature at the present time is full of reports of diagnostic errors wherein other organs are blamed, and removed, when the real pathology lies in the genito-urinary tract.

It is needless to say that the appendix is the chief sufferer, is frequently decapitated, and recorded as a surgical cure, when the painful pathology lies elsewhere.

Next in frequency come the ovaries, whose actual or fancied pathology is made to account for the pain of kidney, ureteral or bladder disease. The gall bladder does not escape, but is also wrongly accused.

These patients, after operation, are partially relieved, probably because of the enforced rest, diet and hygiene coincident with modern surgical methods. They are never absolutely cured. Although the pain because of which they were operated upon may not recur, still there is that lack of complete relief which we regard as proof of correct diagnosis. They drift away from the man who operated upon them, and come to correct diagnosis at the hands of the urologist, or on the autopsy table.

Accidental finding of blood or pus in the urine, or recurrence of pain, sends them to the urologist; or the pathologist finds unlooked for stones or neoplasm at post mortem. The urine may be absolutely negative, but a careful history will first direct attention to the genito-urinary tract. Symptoms may have been present for as long as forty years before a correct diagnosis is arrived at; and it is by no means uncommon to be told that the present complaint has been in evidence for from a few months to seven or eight years.

The honors for fooling the doctor are about equally divided between stone in the kidney or ureter, and pyelonephritis. Renal tuberculosis is next in evidence, to be followed by hydronephrosis and hydro ureter. Tumors of the kidney and congenital malformation may also simulate ab-

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dominal disease. Nephroptosis, probably because it is easily diagnosed by palpation, or, when it is not thus easily found, is better left alone, does not furnish a large factor of error.

There are definite indications which call for a complete urological examination. First and most important of these is real abdominal pain which has remained unrelieved by one or more operations intended to produce relief. As a whole, the profession is becoming more fully aware of the possibilities of lesions of the genito-urinary tract as a source of diagnostic error and in the future this indication will not take first place. However, at the present time, fully fifty per cent of the total cases coming to the urologist for diagnosis have been subjected to one or more unsuccessful s u r g i c a l operations. There is no disease of the abdominal viscera which may not be simulated by a urological condition, and while pain is the most frequent presenting symptom, what has been said of it is true of other conditions, such as vomiting. The presence in the urine of blood or pus, or hemorrhage from the uretha, should also require that the patient be subjected to a complete urological examination. That the profession is not fully aware of the importance of abnormal constituents in the urine is evidenced by recent health surveys in two of our cities, in which there were nearly twice as many x-ray examinations of the chest and Wasserman reactions run as there were routine urinalyses made.

The classical pain of kidney colic or stone most certainly calls for complete urological examination. This classical picture may be produced by blood clots or pus passing down the ureter, without stone being present. Although the eye in the bladder, through the cystoscope, may frequently guess at the pathology present, infection and hemorrhage are not to be differentiated from stone except by catheterizing the ureter, and injecting the pelvis with opaque fluid when necessary. It is only by pyelography that a pedunculated tumor in the kidney pelvis, blocking the ureter and causing colic, is to be diagnosed.

Kidney stones and ureteral stones do not always give x-ray shadows which are characteristic. The uric acid stones cast negative shadows and usually cannot be detected by the roentgenologist. Such stones may pass into the ureter, block it partially or completely, and lapse into silence to be found a variable time afterward by the inquiring nose of the ureter catheter. After sufficient calcium is deposited in or around the stones they become visible on the radiograph (Fig. 1). Shadows simulating ureteral stone may be very close to the ureter area (Fig. 2) and opaque catheter is necessary to determine their relation to the ureter (Fig. 2a). By the injection of an opaque fluid through the catheter which has passed a stone or stricture, the amount of damage done to the ureter and kidney is easily measured with the aid of the x-ray (Figs. 3 and 4). Where one stone has come from, there may be others also, doing kidney damage (Fig. 5). Pyelography serves to determine if the factory is still working by localizing doubtful shadows, which might be stones, in the region of the kidney (Fig. 6), or by accentuating transparent stones with a halo of injected bromide solution.

The patient, as well as the doctor, is frequently satisfied when the pain of ureteral colic has subsided coincident with the passing of the stone into the bladder, where it may remain or be passed spontaneously. If it is not so passed very shortly after it reaches the bladder, it soon becomes too large to enter the urethra and remains as a foreign body in the bladder, where it gives rise to numerous symptoms. Stones in the bladder may be small (Fig. 7), and almost insignificant in symptoms; or they may be very large before they give rise to much trouble (Fig. 8). They are frequently embedded in the bladder mucosa and interfere more or less with the passage of urine. One was

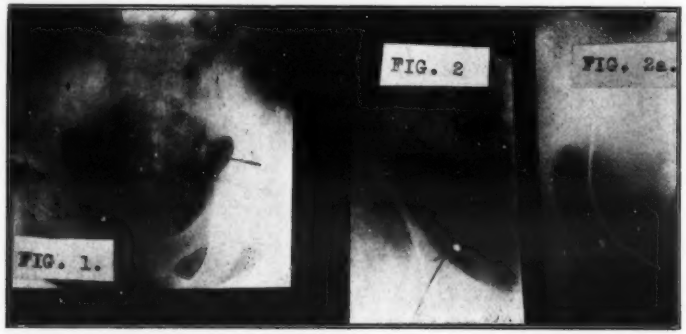


Fig. 1.—Stone in lower ureter of child, producing complete obstruction.
Figs. 2 and 2a.—Shadow suspected of being stone, oriented by opaque catheter in ureter; shadow not in line of ureter.

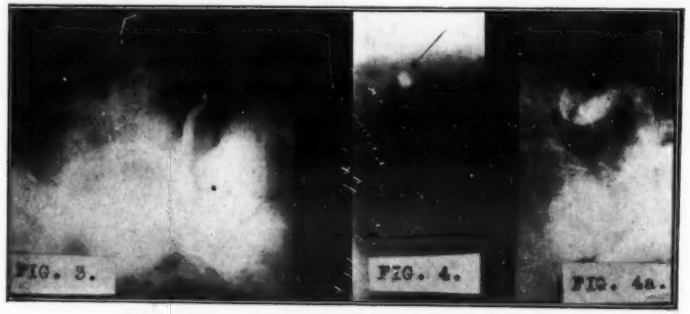


Fig. 3.—Ureteral stone, blocking ureter which is outlined with opaque fluid. There is a stricture at location of stone, a secon stricture higher up, and a stone in kidney pelvis.
Figs. 4 and 4a.—Stone in ureter, near kidney, producing complete obstruction, with hydronephrosis. Stone shown before injection of ureter, and its location indicated by arrow in film made with ureter outlined.

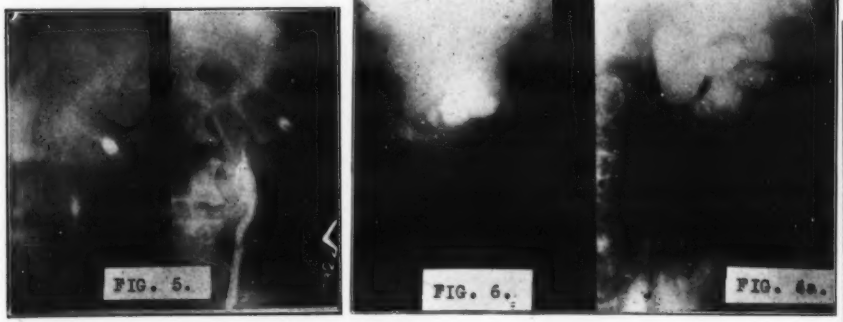


Fig. 5.—Stone in ureter, with a second stone in kidney pelvis, shown before and after injection of ureter.
Figs. 6. and 6a.—Stone in kidney pelvis, proven by injecting kid ney pelvis, the opaque fluid surrounding stone. Kidney has hydronephrotic pelvis.

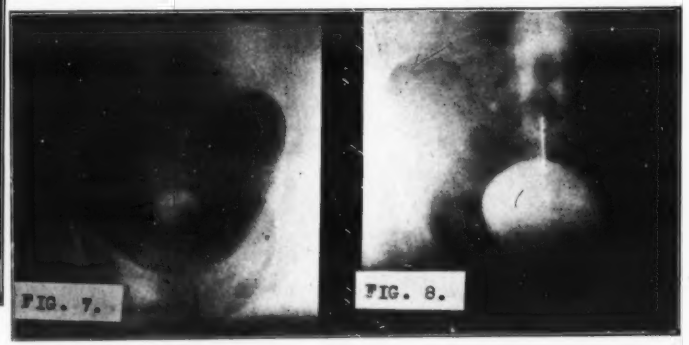


Fig. 7.—Small, round stone in bladder.
Fig. 8.—Large, dense calcium stone in bladder, evidently of long standing but only recently producing symptoms.

found (Fig. 9) having a neck which would, at times, project into the urethra and block the passage. Stone is frequently suspected when a sound passed into the urethra is heard to grate over it or to produce a metallic click when the stone is touched. Nothing short of cystoscopic and radiological examination, however, is adequate to determine the exact size of the stone, and, what is more important, the preferable method of removal. A stone which is too large to crush with lithotrite will interfere considerably with complete cystoscopic examination of the bladder, and it is here that the cystogram is desirable. In doing cystoscopic examination of the bladder, the suspected stone may be highly refractile and must be differentiated from encrusted cystitis. A bladder stone of any size will cast a shadow when the light of the cystoscope is thrown upon it, or it may be turned from side to side with the catheter so that full appreciation of its size and shape may be gained.

Stone is only one of the conditions in the bladder in which radiography is valuable in completing the picture. In planning the surgical approach, a cystogram is frequently necessary to determine if the bladder shall be opened or attacked from the retroperitoneal aspect.

The normal cystogram (Fig. 10) is smooth, without filling defects, and variable in size. If the bladder is irritable, it may contract and permit the comfortable injection of only a small quantity of fluid. External structures may indent the bladder, but this must not be mistaken for intravesical pathology (Fig. 11).

A projection into the bladder of some structure outside it is very easily appreciated by the cystoscope as the normal mucosa of the bladder is seen to rise over the tumor mass.

Diverticuli of the bladder, unless located directly posterior to the organ, usually show very clearly and definitely in the cystogram (Fig. 12). They are single or multiple, and may vary considerably in shape and size. Very large stones in the lower end of the ureter (Fig. 13) occasion the same symptoms as diverticuli, and may not be diagnosed except by cystoscopic examination. Indeed, the combined radiological and cystoscopic examination has its most necessary indication here. By passing the beak of the instrument into the diverticulum it is illuminated, its extent measured, and its contents seen, whether this be stone or pus.

Papillomatous growths of the bladder are to be suspected when the urine contains blood, and are to be thought of very strongly when anything like a profuse hemorrhage takes place from the urethra. Upon examining a bladder containing papilloma, the seaweedlike appearance of the growth is apparent at once. If the bladder is very full of these tumors, introduction of the cystoscope into this will be very like what would be seen if the ocular were pushed into a bunch of grapes; the glistening white surface of the papilloma reflects the cystoscope light and the growth itself may obscure the vision. By drawing the beak of the cystoscope away from the papilloma, a truer appreciation of its extent is obtained.

The cystogram shows filling defects, usually small serrations (Fig. 14) if the growth is along the vault; if on the floor, the cystogram may show nothing.

Cancer of the bladder shows cystographic and cystoscopic peculiarities which are characteristic. The cystogram shows (Fig. 15) filling defects analogous to those found in the barium filled stomach, which contains new growth. While this is true of carcinoma which has attained any considerable size, early diagnosis of carcinoma must be made by the appearance of the mucosa through the cystoscope, and is very difficult at times.

Small amounts of pus, in the voided specimen from the male, are of little significance unless an antecedent gonorrhea is eliminated. Pus in the catheterized specimens in a male who

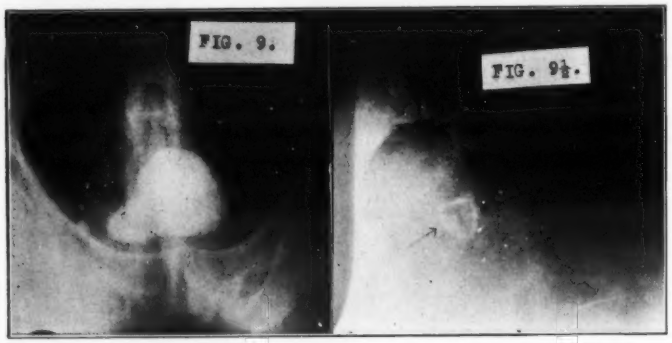


Fig. 9.—Large bladder stone, with conical shape, the small end producing intermittent obstruction by falling into the urethra.
Fig. 9½.—Shadow diagnosed as bladder stone, but which proved, at operation, to be a calcified fibroid in anterior wall of uterus. Shows necessity for cystoscopy in such cases.

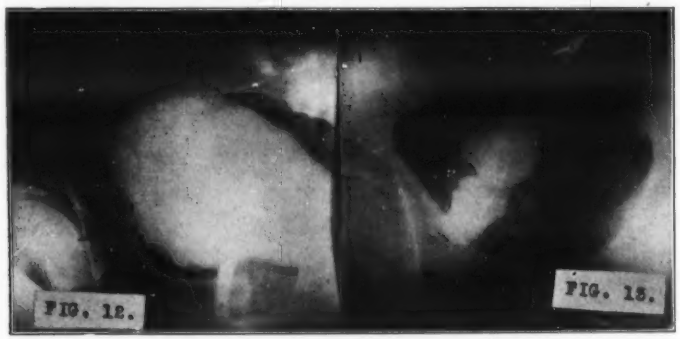


Fig. 12.—Two large diverticuli of the bladder.
Fig. 13.—Group of large stones which were entirely within the lower ureter, not being visible, nor removable, from the inside of the bladder.

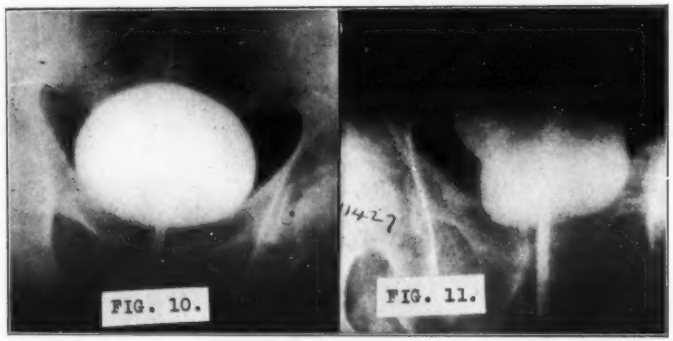


Fig. 10.—Normal, smooth, round contour of cystogram.
Fig. 11.—Normal cystogram, the indentation in the outline being produced by external pressure.

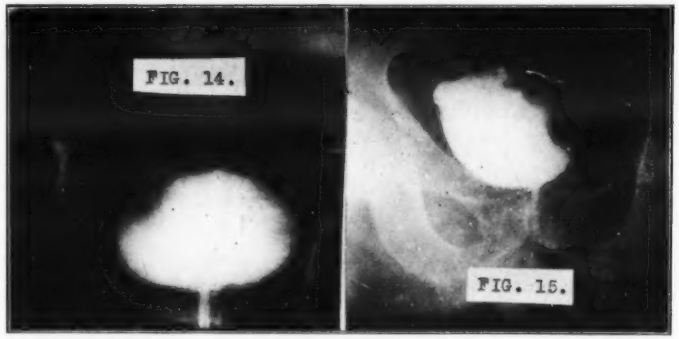


Fig. 14.—Cystogram of a papillomatous bladder; note the serrated outline along the upper surface, being the filling, defects of the papillomatous growths.
Fig. 15.—Cystogram of bladder carcinoma, showing the deformity of outline, the marked filling defects, the absence of shadow where tumor projects into bladder, and the ureter partly filled.

denies gonorrhea is suggestive of a lesion in the kidney or ureter and calls for complete cystoscopic examination. In those cases in which the previous history of gonorrhea is obtained, it is very interesting to demonstrate the location and extent of the urethral damage by opaque fluid. The normal urethra (Fig. 16) is fairly regular in size, and usually dilates when the patient strains, with the meatus closed during radiography. Under these circumstances, stricture of the urethra is nicely shown (Fig. 17) and its degree and extent estimated. Stricture, naturally, must be dealt with before cystoscopy can be successfully carried out.

Having, then, excluded gonorrhea as a source from which pus in the urine comes, we must consider from what other sources it might spring.

Stone in the kidney is usually the first thought of the general practitioner, especially if there is any pain referred to the kidney. While nephrolithiasis is relatively unimportant, as compared with the other lesions of the kidney, it should always be examined for. Unfortunately, many kidney stones do not cast shadows on the radiographic film. Only the calcium stones cast recognizable shadows. The kidney outlines can usually be shown in a properly made radiograph (Fig. 18), and frequently the plain films will show stones—which may be either small (Fig. 19), or quite large (Fig. 20).

The chief danger from kidney stone lies in their tendency to pass into the ureter (Fig. 21), where they may lodge and become a very painful and dangerous condition. The common result of stone which remains in the ureter for any length of time is a hydronephrosis, which is brought about also by the kinking of the ureter, or from infection entirely independent of stone. Outline of the kidney pelvis and ureter by opaque media is absolutely necessary to the fullest appreciation of the extent of the hydronephrosis. In order to understand the variations produced in the pyelograms of hydronephrosis, it is necessary to appreciate the normal kidney pelvis, which varies considerably in size as well as shape.

The normal kidney pelvis may occur in several forms, the chief of which are the following: (1) The typical pelvis, which has only an upper and lower calyx (Fig. 22). (2) The branched pelvis (Fig. 23), where in addition to the upper and lower calyces there is a middle calyx; this middle calyx may empty either directly into the pelvis or may join one of the other calyces, usually the lower. (3) The hemipelvis (Fig. 24), in which the lower calyx is large, practically taking the place of the pelvis, receiving several secondary calyces, and the upper calyx is poorly developed, sometimes joining the lower by a narrow passage. (4) The ampullar type (Fig. 25) of pelvis is one in which there are no major calyces, but a number of minor calyces entering directly into a somewhat rounded and roomy pelvis. (5) The infantile type is a small, undeveloped pelvis, practically a continuation of the ureter (Fig. 26). (6) A rare type is that in which there is a principal pelvis which communicates with secondary pelves, which latter, in turn, communicate with the major calyces.

The pelvis may hold from three to eight c. c. of fluid, with ten c. c. as the extreme normal limit. It is usually roughly pyramidal in shape, but its shadow may appear somewhat rounded, if any pressure is exerted in the injection of fluid. The calyces should be well outlined. The ureter varies much in size, depending also on the amount of pressure exerted.

The pyelographic evidence of hydronephrosis (Figs. 27-32) is a dilatation of the pelvis and calyces, the latter being enlarged, and rounded instead of being pointed or serrated. The cause of the hydronephrosis is ureteral obstruction, whether due to simple kinking, to stone or to stricture. Kinking of the ureter usually occurs in the upper three or four inches. This may result in nothing more than critical pains, and when

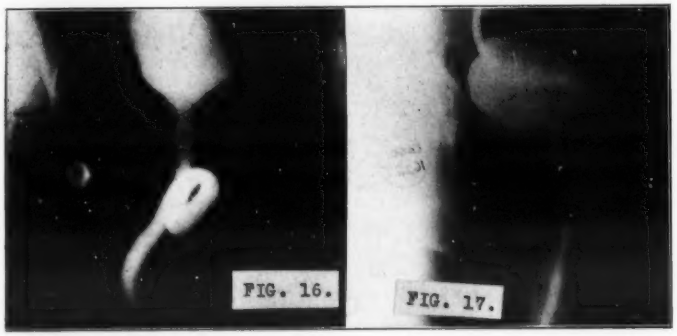


Fig. 16.—Outline of a large, normal urethra, filled by the patient attempting to void the opaque fluid injected into t he bladder, while external meatus is held closed.

Fig. 17.—Stricture of the urethra just posterior to bulb, extent of which is outlined by opaque fluid injected into the bladder and forcible attempt to void made, while meatus is held closed.

Fig. 18.—Normal radiographic outline of kidney.

Figs. 19 and 19a.—Small kidney stone, shown to be in lower pole of kidney, by injecting pelvis, for orientation.

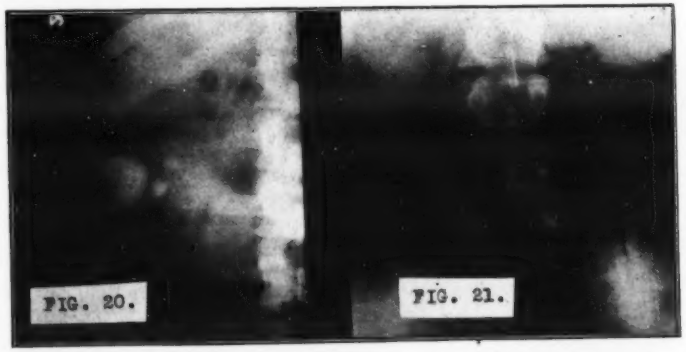


Fig. 20.—Group of large kidney stones, producing very mild symptoms.

Fig. 21.—Ureteral stone shortly after its passage into ureter. Patient was having ureteral colic. Stone passed into bladder about 48 hours later.

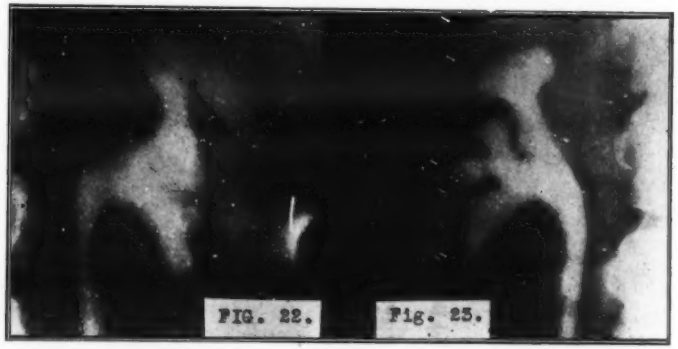


Fig. 22.—Normal Type I kidney pelvis, with upper and lower calyces.

Fig. 23.—Normal Type II kidney pelvis, with upper, lower and middle calcyces. The middle calyx may join the upper or the lower calyx, or may enter directly into the pelvis.

due to simple mobility of the kidney may not be sufficiently continuous or permanent to produce dilatation of the pelvis and hydronephrosis.

Stricture of the ureter is caused by scar tissue as the result of trauma from stone, or as the result of infection. The degree of hydronephrosis will depend on the length of time it has existed and the completeness of the stricture.

Pyonephrosis, which results as a sequence of hydronephrosis and is, therefore, dependent upon the same causes, is not to be diagnosed from hydronephrosis by the x-ray, but is proven to be present when the return through the ureter catheter is purulent.

Tuberculosis of the kidney is to be suspected whenever the urine contains blood or pus. Radiographic methods are of less value than cystoscopic examination, although calcifications of old tuberculosis are sometimes found (Fig. 33), and when cavities are formed these may communicate with the pelvis and be filled by the opaque fliud in pyelography (Figs. 34 and 35). Slight variations from the normal cystoscopic appearance require further examination. Early in kidney tuberculosis there is produced an area of redness and edema around the ureteral orifice. If nephrectomy is done, this redness and edema will subside. Later actual infection of the bladder with the formation of tubercles occurs. It is of the utmost importance to determine whether the bladder tuberculosis is secondary to a renal lesion or to genital tuberculosis, particularly in the male. The finding of tubercle bacilli in the urine secured through ureter catheter is conclusive evidence that the kidney is tuberculous.

It is beside the scope of this paper to enter into the diagnosis of lesions in the genital tract except to say that they should be excluded. It is very important to be sure that both kidneys are not tuberculous before proceeding to remove one of them.

Having excluded kidney tuberculosis and stone as the source of blood, with or without pus, in the urine, tumor of the kidney should be suspected. Diagnosis of kidney tumor during the operable stage taxes all the ability of the urologist. Spontaneous hemorrhage through the urethra, if the bladder does not contain new growth, should always arouse suspicion of kidney tumor. Formerly the presence of tumor cells in the urine was considered the best evidence of new growth, but this method is no longer regarded as reliable. It has recently been said that trauma of the kidney pelvis by means of the ureteral catheter, if it produces bleeding, is diagnostic of tumor. However, bleeding from a normal pelvis is easily produced by a catheter introduced too far, and should not be considered pathognomonic of tumor. Plain radiographs of the abdomen, properly taken, will usually show the kidney outline, and the outline may be so normal in position and size as to rule out gross tumor (Fig. 18). If there are variations in the normal uninjected kidney outline, or if there are irregularities in the shadow of the kidney pelvis on the pyelogram, these are strongly suggestive of tumor. If the kidney tumor is large, it will usually displace the pelvis (Fig. 36). Should the usual pyelographic methods fail, or if the ureter is blocked so that the catheter cannot be introduced, the kidney may be beautifully shown by pneumoperitoneum or perirenal emphysema. In pneumoperitoneum the peritoneal cavity is inflated with a gaseous medium (oxygen, carbon dioxide or plain air), and the kidney examined under the fluoroscopic screen or by radiographs. In perirenal emphysema, the fatty bed of the kidney is infiltrated with oxygen until the kidney stands out in a bed of gas, its contour being plainly visible by x-ray methods.

Illustrating the methods outlined, the procedure pursued in a recent case referred because of spontaneous brisk hemorrhage through the urethra, is outlined. Normal cystogram was obtained (Fig. 37). Cystoscopic

Fig. 24.—Normal Type III kidney pelvis, the lower calyx being well developed and practically takes the place of the pelvis, while the upper calyx is poorly developed.

Fig. 25.—Normal Type IV kidney pelvis,—the ampullar type, with large, pyramidal pelvis, receiving several calyces directly. Must not be mistaken for beginning hydronephrosis.

Fig. 26. Normal Type V kidney pelvis. The infantile or undeveloped type, which appears like an extended ureter, branching directly into calcyces. It is usually of small volume and can easily be overdistended.

Fig. 27.—Beginning enlargement of a kidney pelvis of Type III shape, produced by contracture of ureter below. There is rounding of the calcyces, slight increase in capacity, and the ureteral stricture was demonstrable under the fluoroscopic screen.

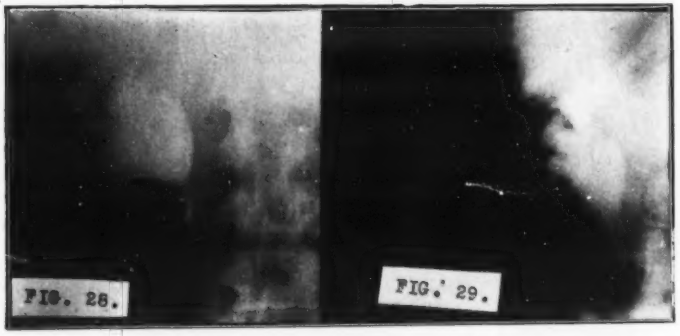


Fig. 28. —Fairly well advanced hydronephrosis from stricture of the ureter shown just below the pelvis.

Fig. 29.—Hydronephrosis from ureteral stricture low down; pelvis markedly dilated and rounded.

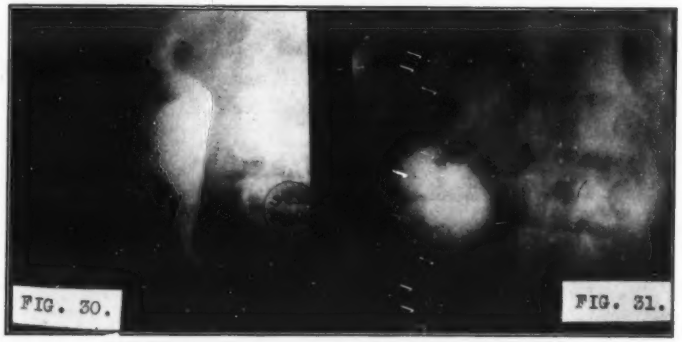
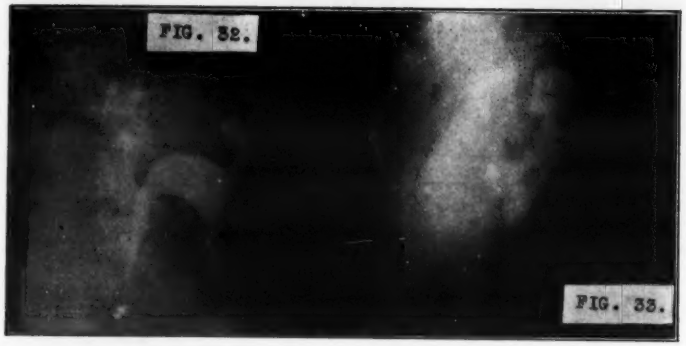


Fig. 30.—Moderate degree of hydronephrosis from ureteral obstruction.

Fig. 31.—Pyonephrosis, following ureteral obstruction, produced by stone. Has the same characteristics as hydronephrosis.



Figg. 32.—Hydronephrosis of moderate degree, produced by ureteral kinking from loose kidney. Note position of kidney pelvis.
Fig. 33.—Calcifications in kidney from tuberculosis.

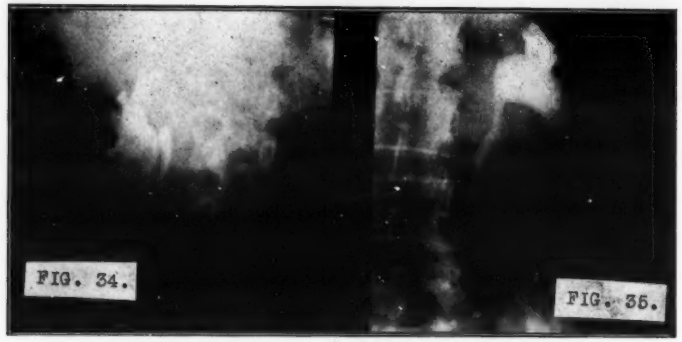


Fig. 34.—Extrapelvic shadow, probably the pocket of an old tuberculous abscess which has opened into the kidney pelvis.
Fig. 35.—Tuberculous kidney, with a small pocket communicating with the pelvis. Sharp kink in ureter near pelvis.

Fig. 36.—Displacement of pelvis from enlargement of kidney (tumor?). Note the direction and point of entrance of catheter into the kidney pelvis. Marked hydronephrosis.
Fig. 37.—Normal cystogram in a case of kidney tumor.

Fig. 38.—Normal Type III pelvis in same case as Fig. 37.

examination showed a small trickle of blood emerging from the left ureteral orifice. The right ureter was found and catheterized, and normal kidney pelvis outline shown by pyelogram (Fig. 38). An obstruction or

Fig. 39.—Lateral view of abdomen of patient with peritoneal cavity inflated with oxygen (pneumoperitoneum); patient is supported with pendant abdomen, gas outlining the prevertebral spaces and showing outline of much enlarged kidney on left side.

Fig. 40.—Antero-posterior view of same patient as Figs. 37-38, with pneumoperitoneum. Liver, spleen and both kidneys outlined; left kidney much enlarged, extending down to pelvic brim.

obliteration of the left ureter was located about one-half inch from the orifice; this obstruction resisted two attempts at catheterization. Indefinite shadow in the left kidney region suggested tumor, and pneumoperitoneum was resorted to, followed by x-ray. After inflation of the peritoneal cavity with oxygen, the patient was turned in the prone position,

Fig. 41.—Double ureter, with two separate kidneys on the same side, with a different excretion time for phthalein, showing them to be separate structures entirely.

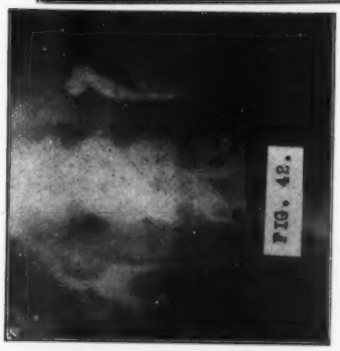


Fig. 42.—Ureter branching at its entrance to the pelvis, the upper branch passing to a separate pelvis.

Fig. 43.—Double ureter, with two separate pelves, one of which is markedly hydronephrotic, the other being of normal size and contour, though poorly developed.

raised upon blocks until the abdomen was pendulous, and the gas had risen into the prevertebral space. Tumor mass was easily seen extending from the diaphragm to the pelvic brim (Fig. 39); antero-posterior examination showed the tumor even better (Fig. 40). A probable diagnosis of hypernephroma or sarcoma was made.

In routine pyelographic work, interesting and unexpected findings are frequently encountered. Branching ureters with double kidney pelvis may be discovered (Fig. 41). A double kidney is less frequently found; one was recently discovered (Fig. 42) in which the phthalein appeared in the urine from the upper pelvis in five minutes and from the lower one in twelve minutes, showing that the blood supply and kidney structures were entirely distinct. In another case, one kidney pelvis showed marked pathology of hydronephrosis, while the other pelvis on the same side was practically normal (Fig. 43).

In conclusion, yet us urge you to be on the lookout for genito-urinary pathology, especially in doubtful abdominal conditions, and to make a practice of routine urinalysis in every case which presents itself to you for diagnosis, no matter how obvious the diagnosis may appear to be. Should there be any suspicion of pathology in the urinary tract, a thorough and comprehensive examination is indicated, utilizing the methods which have become thoroughly established as indispensable in urologic investigation.

CHRONIC AND SUBACUTE APPENDIX DISEASE*

W. WARNER WATKINS, M. D., F. A. C. P., Pathological Laboratory, Phoenix, Arizona.

The late John B. Murphy prefaced one of his clinical lectures on appendicitis with the statement that, as long as the hospital mortality from operated acute appendicitis still approaches ten per cent, the subject will be a live one for discussion.

With regard to chronic appendicitis, as long as the subject remains the battle ground for opinions as widely divergent as human ideas can be, we may study the subject with profit and entertainment.

Reginald Fitz, of the Massachusetts General Hospital, introduced the term "Appendicitis" in 1886, and the medical world still wonders why someone did not conceive the idea earlier. Yet twenty years more passed before a comprehensive description of chronic appendicitis appeared (Klemm in 1906), and, today, in the same hospital, one of the greatest clinicians of this generation denies the existence of such a clinical condition as chronic appendicitis.

Richard Cabot[1] is the best-known exponent of the absolute negative in the gradations of medical opinion with regard to chronic appendix disease. He claims that, in his enormous experience, he has never yet seen a patient whose symptoms he could attribute to a chronic disease of the appendix. In his work on "Differential Diagnosis" he tabulates 15,000 cases of "chronic dyspepsia", in 12,600 of whom the gastric symptoms were due to conditions outside of the stomach; among such non-gastric conditions he does not list chronic appendix disease even as a possibility.

The literature on chronic appendix disease is enormous, there being three or four hundred articles in American journals alone, and time will permit only a mention of a few of the authorities. Among the writers whose views approach those of Cabot, without being quite so extreme, are Bevan[2], Williams & Slater[3] and Con-

*Read before the New Mexico Medical Society, at Gallup, N. M., April 30, 1922.

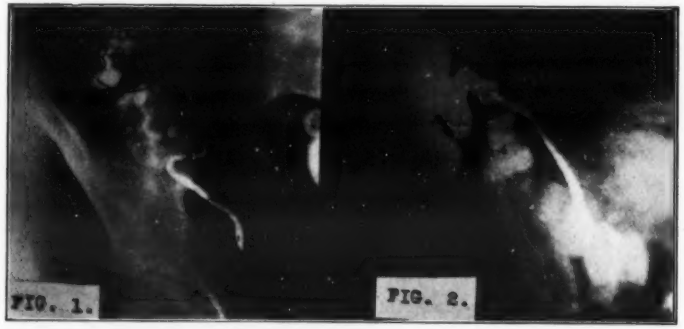


Fig. 1.—Normal appendix, at 24 hour period, freely movable, without tenderness or other symptoms.
Fig. 2.—Normal appendix, at six hour period; freely movable, not sensitive or tender.

Fig. 3.—Retrocecal appendix in child, at 24 hour period; cecum nearly empty.
Fig. 4.—Short, blunt, retrocecal appendix, which was tender and painful; found in subacute stage at operation.

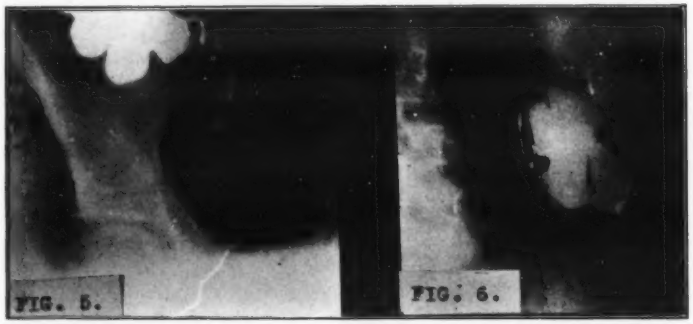


Fig. 5.—Long, coiled appendix, with slight filling defects; otherwise normal.
Fig. 6.—Short, kinked appendix, turned up and adherent to ileocecal valve region.

Fig. 7.—Long, slender appendix, turned upward behind and to outer side of cecum; adherent and tender.
Fig. 8. Appendix in stage of acute inflammation; short stiff and very tender; lumen imperfectly outlined, because of being already filled with pus (shown at operation).

Fig. 9—Appendix coiled up in a mass of adhesions; very tender and painful; operation showed subacute infection.
Fig. 10.—Appendix adherent to cecum through half its length, with constricted areas, concretion defects, and showing definite tenderness to palpation.

Fig. 11.—Appendix adherent to posterior abdominal wall, shown on stretch, with two concretion filling defects. Symptoms suggested stomach ulcer.
Fig. 12.—Appendix kinked and adherent, with concretion defects in tip. Symptoms typical of stomach ulcer, including several hemorrhages from stomach. When operated during an acute appendicitis attack, stomach was found normal, and all ulcer symptoms disappeared after operation.

Fig. 13. A very long, imperfectly filled appendix, adherent in right side of pelvis, and very sensitive. Operation showed adhesion to right ovary and subacute infection.
Fig. 14.—A long appendix, adherent to head of cecum, showing several strictures and filling defects of concretions; tender and painful.

Fig. 15.—Cecum on floor of pelvis, with appendix firmly adherent to some structure in left pelvis; shardly kinked at the tip. Left sided pain.
Fig. 16.—Appendix turned upward and adherent to the transverse colon, deforming the cecum. Pain, tenderness, and stasis.

Fig. 17.—Appendix adherent along the inner border of cecum; irregularities in contour; definitely tender.
Fig. 18.—Appendix which shows an extra-appendiceal barium shadow, which was diagnosed a perforation. It proved to be so at operation, showing a walled off pocket at the point indicated.

Fig. 21.—Very irregular filling of appendix lumen, with constricted points and concretion areas. Very tender.
Fig. 22.—Long, sensitive appendix, with two definite concretion defects distal to basal contraction of lumen.

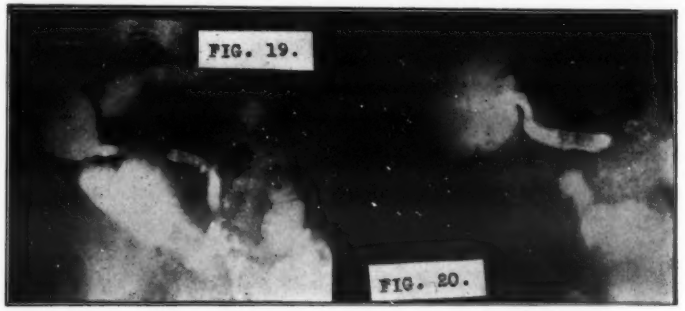


Fig. 19.—A very long appendix, at the six hour period. Shows several areas of irregular filling. It was movable, but was very tender. Clinical symptoms were gastric entirely.
Fig. 20.—Appendix with concretion in distal half, and constriction near base. Definitely tender. Clinical symptoms were gastric and suggested duodenal ulcer.

Fig. 23.—Appendix showing a filling defect which could be definitely diagnosed an ulceration in the appendix. Such areas may occur around fecaliths in the appendix.
Fig. 24.—A segmented appendix, with three sacculations filled with barium, and two large areas of contracture. Such an appendix, when tender and accompanied by clinical symptoms, is evidently pathological and probably infected at the time.

Fig. 25.—Appendix with concretions in the distal half and constricted proximal lumen. Tender and painful with reflex gastric symptoms.

Fig. 26.—A very long, irregularly filled appendix, with multiple constrictions and concretions. Adherent in pelvis (right tube at operation), tender and painful.

Fig. 29.—Tuberculous appendix, showing irregularities in outline. A short, blunt, stiff, tender appendix. Operative confirmation.

Fig. 30.—Tuberculous appendix; short, blunt, stiff and irregular in outline. Operative confirmation.

Fig. 27.—Appendix adherent opposite lower sacro-iliac joint. Much constricted in proximal two thirds, with large sacculation and concretion in tip. Very sensitive. Found adherent to bladder at operation.

Fig. 28.—Very long appendix, extending into pelvis, but not adherent. Multiple concretions in distal half; Much constricted in proximal portion. Tender. Patient also had doudenal ulcer.

Fig. 31.—Tuberculosis of appendix and cecum. Taken at six hour interval, showing the hypermotility of cecum and intolerance to barium meal. Appendix kinked, adherent, and very sensitive.

Fig. 32.—Retrocecal appendix, probably tuberculous, showing irregularities in filling, with hypersensitive cecum, such as is found in tuberculosis of this part of the bowel.

nell[4]. At the opposite extreme are those who claim that any pathological change in the appendix, whether fibrosis, obliteration, adhesions, or inflammation, is an emergency demanding surgery. The midground, and probably the correct ground, is that held by such writers as Deaver[5], Mayo[6], Mohnihan[7], Horsley[8], Brewer[9], Graham[10], Stanton[11], Lichty[12], Cheney[13], Bassler[14], Brown[15], Aaron[16], and others. These recognize chronic appendix disease as a frequent cause of gastrointestinal symptoms, and recommend surgical relief, after comprehensive diagnosis.

With regard to the morbid anatomy of appendicitis, whether acute or chronic, we are on fairly uncontested territory, the work of Moschkowitz[17] and MacCarty[18] being generally accepted. The divergence comes on the question as to whether the pathological changes which are so frequently found are the cause of reflex symptoms. For our own satisfaction and instruction, the pathological work of Moschkowitz has been confirmed by my associate, Dr. H. P. Mills, on a series of appendices now numbering about 500. Our conclusions have not changed since Dr. Mills' pathological report two years ago[19], and may be summarized as follows:

(1) Pathologically considered, chronic appendix disease is an end result and not an active process. When attacks of acute or subacute appendicitis subside, they leave sequellae of various sorts—fibrosis, strictures, kinks, adhesions. These, while not dangerous in themselves, may excite motor phenomena and reflex symptoms in the stomach and colon, or lead to recurring attacks of subacute appendix infection.

(2) It is immaterial, from the standpoint of the evolution of chronic pathological changes in the appendix, whether these recurring or primary infections come by way of the bowel lumen or through the blood stream. In either event, the first effect is upon the mucosa of the appendix, which becomes swollen and infiltrated with leucocytes; should the inflammation subside at this point regeneration may be complete without permanent damage. If inflammation continues beyond the first twelve hours, there will be a round cell infiltration of the submucosa and muscularis, with a peritoneal exudate; subsidence of the inflammation at this point is followed by a varying degree of fibrous tissue formation, thickening of the appendix wall and narrowing of its lumen; the peritoneal exudate, if slight, may disappear, or it may organize with adhesion of the appendix to some contiguous structure. The third stage of acute appendicitis is represented by deeper necrosis of the mucosa and submucosa, pus formation, more extensive peritoneal exudate, perforation or gangrene. The second stage inflammations, resulting in some degree of permanent damage and deformity, are those which concern us.

(3) Should the inflammation affect one segment of the appendix more than another, the final healing fibrosis will result in strictures and an irregular lumen. Distal to such strictures or between two of them there will be sacculations in which fecal contents may be trapped until they harden and concretions form; these may then act as ball valves to keep the appendix from emptying properly. Attempts to empty will result in cramplike pains and reflex motor phenomena in the stomach and colon. Furthermore, the trapped contents invite the transformation of intestinal bacteria into pathogenic forms, or permit the development of a few incidental already virulent bacteria into a sufficient number to produce local or metastatic infection. Thus we will have recurring attacks of subacute or acute appendicitis which still further deforms the appendix and a vicious circle is established.

(4) The peritoneal exudate, when organized, may adhere the appendix to some neighboring structure, with varying effects upon the appendix itself or the organ to which it is adherent.

To deny the actuality and importance of chronic appendix disease is to deny that these admitted lesions and abnormalities are serious. What are we to say about an appendix which becomes inflamed, heals, and is left adherent to the right ovary, or turned up and adherent to the gall-bladder, or left with a constriction in the middle and fecal concretions in the distal half, or sharply kinked so that it cannot empty its contents? We believe that chronic appendix diseose or, if the term is preferred, "recurrent subacute appendicitis", is the most frequent form of pathology producing what the patient will call "stomach trouble." In a recent report[20] of one thousand consecutive examinations of the gastro-intestinal tract in patients presenting symptoms of abdominal disease, 323, or 32 per cent, showed appendices demonstrably pathological in the x-ray examination. In 212 of these patients, the appendix pathology was the only lesion which could be found. In 25 additional cases there were ileocecal adhesions, without the visualization of the appendix, in which the adhesions were doubtless due to healing of the former appendix infection.

The SYMPTOMS which a chronic or subacute appendix disease will produce vary as widely as the pathology.

The pain and discomfort will usually have some relation to the position of the appendix, and the presence of adhesions. The pain may be a burning sensation in either hypochrondrium, or in the epigastrium; it may occur after eating or before eating, and may be accompanied by nausea, belching or distention. There may be a complete remission of symptoms for weeks, thereby resembling gall-bladder disease. There may be a reflex hyperchlorhydria, or gastric hemorrhage, as in the cases described by Outland & Clendenning[21], and seen by us in at least one case. The pain may be localized in the right iliac region, may be diffused, or may occur on the left side; may be constant or periodical, and the patient will usually describe it as an annoying sense of discomfort. The appetite will usually be capricious, and the general symptoms may be fatigue, headache, failure to gain weight, etc. The urinary symptoms, especially in retrocecal appendices, are important. They resemble urinary tract stone very closely, backache, pain on urination, cramplike pains in abdomen, or even hematura, as reported by Speed[22].

Constipation is very common, due to the disturbed motor functions of the colon, resulting in atony, stasis and colitis. Diarrhea is an occasional result, instead of constipation.

There are usually definite physical signs, but owing to the change in position of the appendix and the pull of adhesions when palpated, the tenderness may be in very unusual locations. Palpation of the appendix region may cause epigastric distress from pylorospasm.

Whenever a radiologic examination is possible, this should be utilized, because it is the most positive method of determining the pathological conditions present and arriving at a diagnosis. In the x-ray examination, what is the evidence of appendix disease? Bearing in mind the evolution of disease changes in the appendix, when this is chronically or intermittently infected, it is necessary to demonstrate gross pathology either under the fluoroscopic or on radiographs. In all the 300 cases cited, there was such gross and visible pathology.

TECHNIC: The stomach, with a barium meal, is always inspected to determine the presence of reflex motor phenomena; then at the end of six hours, the ileo-cecal region is examined on fluoroscopic table for tenderness, mobility and filling defects. Usually at the end of twenty-four hours, sometimes at twelve or eighteen hours, the same region is again examined. If the appendix is visualized, it is inspected under direct palpation for its position, for adhesions, for filling defects, and for tenderness. (Normal Fig. 1-2).

(Figs. 3-7) POSITION: The appendix may be congenitally retrocecal or otherwise abnormally placed, and when so located is more likely to give trouble. The appendix may be held in abnormal positions from past disease. The position of the appendix, while not, in itself, of great importance as indicating disease, should be a matter of concern to the surgeon, and should be determined by x-ray before any interval operation.

ADHESIONS: Since one of the earliest effects of an acute infection of the appendix (Fig. 8) is a peritoneal exudate, the most frequent sequellae of unoperated cases are adhesions between appendix and some contiguous structure. Thus we may find the appendix adherent to the gall-bladder, to the right ovary, to the posterior abdominal wall, to the cecum, to the ileum, to the pelvic floor, etc. The appendix may be deformed and kinked by these adhesions. (Figs. 9-17.)

FILLING DEFECTS: It is safe to assume that for every case of acute appendicitis which goes promptly to operation and relief, there are ten which run their course and subside, without operation. These ten, however, may vary in extent and severity from a fleeting infection of the mucosa or submucosa which subsides without marked symptoms, and *usually without pain*, to a frank acute appendicitis, which, for some reason, is not operated. Every attack, whether mild or severe, will leave changes in the appendix If the infection is localized, such as occurs immediately surrounding a temporary fecalith, there will be local destruction of the mucosa, with sequellae varying from slight fibrous tissue formation and constriction at this point, to perforation. (Fig. 18.) If the infection extends throughout the appendix and the entire mucosa is destroyed, the final result will be an obliteration of the lumen. There may be a number of strictures in the appendix from past acute attacks. There may be permanent collections of hard feces in the tip of appendix or in intermediate sacculations. Both constrictions and fecaliths, as well as ulcerations, will show filling defects in the appendix shadow. All three of these abnormalities can be readily distinguished by the x-ray examination, and constitute definite and positive proof of past disease, although they may not always indicate active infection at the time. (Figs. 19-28.)

TENDERNESS: The surgeon considers the finding of appendiceal tenderness significant. It is vastly more significant when found by the radiologist, because it can be determined just what manipulation of the appendix produces the tenderness, whether it is the pull of an adhesion, whether it is direct pressure upon the appendix, or whether the tenderness is associated with contiguous organs, such as the pelvic organs in women. Whatever the other findings may be, tenderness should always be elicited before a diagnosis of active appendix disease is made.

The tuberculous appendix may sometimes be detected by its radiographic appearance when filled with barium. (Figs. 30-32.) The characteristics are the irregular, serrated filling defects, usually a short, blunt shadow, lack of mobility and the tenderness to palpation. The appendix may be tuberculous, and the cecum not yet involved, but the reverse is more likely to be true.

The finding of a demonstrably diseased appendix should not, by any means, deter one from completing the examination of the gastro-intestinal tract. So frequently is appendix disease associated with gall-bladder involvement that there is high authority for the statement that gall bladder-appendix infection is a disease entity and always co-exist. Whether this is true or not, it is certain that they are frequently present at the same time. Furthermore, there is a frequent association between appendix disease and ulcer of the stomach or duodenum.

After it has been definitely determined, in a given patient, that definite pathology exists in the appendix, this does not, by any means, in-

dicate that the patient should be taken to the hospital and operated upon. The clinician or surgeon still has several very important things to decide.

(1) Remembering that the gross changes which the radiologist will demonstrate are, usually, sequellae of past infections, there still remains to be settled the question whether the symptoms now troubling the patient are due to active present disease, to recurrent attacks of infection, to reflex motor or functional disturbances from structural changes in the appendix, or to something else besides the appendix condition. Here comes in the necessity for completing the examination, and not taking too much for granted.

(2) If the further examination reveals associated lesions in the gastro-intestinal tract, it must be decided what relation they bear to the symptoms troubling the patient and whether they are remediable, along with the appendix, by the proposed surgery. Or, on the other hand, whether their relation to the appendix disease is so close that they will recover, when the appendix, as the chief offender, is removed.

(3) If the appendix disease is associated with conditions outside the gastro-intestinal tract which will continue to give symptoms, the operation is likely to be disappointing to the patient, and this possibility should be determined, if possible, before any surgery is attempted.

(4) A long-standing appendix disease will usually be accompanied by well-established structural and functional changes, such as atony of the colon, ptosis of the cecum, neuroses of the stomach, and the possible persistence of these must be considered; the symptoms which the patient will complain of most are usually due to these secondary effects, and failure to relieve them will spell a failure of the operation in the mind of the patient.

(5) A long period of post-operative regimen and medical treatment is usually required, and if both surgeon and patient are not prepared to carry out this regimen, surgery had usually better not be attempted in chronic appendix disease.

In other words, the solution of the problem of chronic appendix disease depends on an accurate and comprehensive diagnosis of the whole pathology present, followed by careful and painstaking surgical and medical treatment, which usually must be carried into months and years after the patient leaves the hospital.

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DEVELOPMENT, RE-ESTABLISHMENT, AND MAINTENANCE OF BREAST MILK*

EARL MENDUM TARR, M. D., Phoenix, Arizona

Human milk is the only ideal food for an infant. This is one fact which needs no comment. It is universally accepted.

Acknowledging this fact and knowing that less than one-third of all infants born are receiving human milk at the end of their sixth month simply means that for some reason, good or bad, they are being cheated. Many factors can and must be considered in solving this problem, but the two paramount things for us to keep before us are: (1) Developing the breast milk in every instance; (2) keeping it up to meet the needs of the individual infant.

To develop the milk of the cow you feed her good food at regular intervals, see that she is comfortably housed or pastured and then milk her dry at each milking. This is exactly what must be done to develop good milk and plenty of it in the human. To keep the cow from "drying up" you continue to feed her a well-balanced ration, you milk her regularly and strip her DRY at each milking. And this is precisely what should be done to keep up a normal and adequate flow of satisfactory breast milk.

For the past five years I have endeavored to discover just why some breasts behave so much better than others. Of ten healthy mothers, two will have an abundance of milk up to the sixth or eighth month; five will have plenty for three or four months and the other three will begin to dry up in six or eight weeks. Careful and repeated observation of the mothers and of the infants, while at the breast, eventually gave me a clue which has helped solve this particular question.

No two breasts are alike and so much may be said of infants. The ducts and ampullae of one breast are unlike those of its mate and the "sucking pad" in one cheek of the infant is scarcely, if ever, the same size of its mate in the other cheek.

My observations have led me to believe that:

1. Infants nurse until they are tired. Then they go to sleep.
2. The amount of milk extracted from a breast by the infant depends upon the size and shape of the ampulla and milk ducts and the development of the sucking pads in the infant.
3. I have found NO INFANT whose sucking pads at birth, or up to the fourth month, were sufficiently developed to completely empty the breast of the mother. The infant becomes tired and goes to sleep long before the breast has been emptied.

With these data before me I have attempted to develop, re-establish and maintain the supply of breast milk by applying rational stimulation to the breast at regular intervals. Mothers are instructed to nurse the infant at one breast for a definite length of time (by the clock), place him back in his crib and then STRIP THE BREAST UNTIL NO MORE MILK CAN BE OBTAINED. This may require ten minutes or thirty.

This added stimulation has, in every instance, resulted in a remarkable increase in the amount of milk and a conscientious mother can keep her breast milk coming almost indefinitely.

TECHNIC OF STRIPPING

Several lengthy articles have been written in which various technics have been described. In 1918 I advised five steps in the technic. Sedgwick, Moore, and Reiss give three movements, or more, and explain them in detail. In a very few simple words the method used by most pediatrists, may be described as follows: Support the breast with the second, ring and little fingers. Place the index finger just back of the

*Read before the annual meeting of the Arizona State Medical Association at Prescott, Arizona, June 14-16, 1922.

areola, below, and the thumb at the upper margin of the areola on top of the breast. Now make a milking (stripping) motion downward and outward to the end of the nipple. This is practically what is now said to be the proper manner in which the breast is to be stripped. Massage of the gland itself should NOT be practiced. Most all women are able after a little practice to satisfactorily empty the breast without causing any pain. If the process of stripping is painful, the technic is faulty.

For convenience of presenting the facts in the cases to be discussed, I have divided them into three groups as follows:

GROUP 1. Mothers who begin to have insufficient milk as early as the fourth to the sixth week.

GROUP 2. Mothers who have, as early as the eighth week, lost their milk.

GROUP 3. Mothers who, at the third or fourth month, find that their milk is rapidly diminishing.

In Group 1, the story is usually the same. A healthy woman has had one or more children and was unable to supply any of them with breast milk for more than three or four weeks. Her present difficulty is that her milk is leaving, the infant has lost weight, cries much of the time and had few and scanty stools. The accompanying history is typical of this group.

Mrs. A. H. T., Albuquerque, New Mexico. Family history and personal history negative. Para 3, one child died at age of 5 months of bowel trouble; second child died when 8½ months old of meningitis. The present infant was born at term after an uneventful and easy labor and weighed 7 pounds 4 ounces. The mother stated that the lying-in period covered ten days and that on the twenty-first day post-partum she had resumed her usual household duties. The infant was being nursed both breasts about every three hours, and when first seen weighed 6 pounds 14 ounces, six ounces less than at birth, and he was then 39 days old. He was brought to me because he cried almost constantly, was not gaining, had very small, hard stools, and seemed really ill. The mother had been indoors practically every day since the babe was born, had taken no recreation, was nervous most of the time and became hysterical upon the slightest provocation.

Examination was perfectly negative except for evidence of starvation. The breasts of the mother were found to be flabby and small, but the nipples were well developed. At 11 a. m. the infant was put to the left breast. He nursed eagerly for 15 minutes and went to sleep. It was found that he got only .6 of an ounce of milk. At 2 p. m. he was put to the right breast. Again he worked eagerly for 15 minutes and then cried for 10 minutes before going to sleep. It was found that at this nursing he got .8 of an ounce. A careful record was kept by the nurse and in the following 24 hours he nursed 7 times and got a total of 3¼ ounces of milk.

On the following day the case was turned over to my special nurse and the usual breast stripping begun. The patience of the mother and the wise and kindly efforts of the nurse resulted in a satisfactory gain as is shown by Chart I.

CHART I. DEVELOPMENT OF BREAST MILK.

Date 1920	Age Days	Weight Pounds	Milk Secretion in ozs.	Totals ozs.
February 16	40	6 14/16	Hours 6 9 12 3 6 10 2	
			By child 1 8 6 4 6 1	1—5 2/5
			By hand 4 4 8 8 4 4	1—4 1/5
			Total in 24 hours, 9 3/5 ozs.	
March 18	70	11 4/16	By child 3 2 2 3 2 2	4—18
			By hand 4 3 3 2 1 2	3—18
			Total in 24 hours, 36 ozs.	

In Group 2 the greatest problem is met. Most of these women, who lose their milk as early as the eighth week, are overworked, undernourished, and are physically and mentally unable to be more than AVERAGE mothers unless careful and well balanced consideration is given them. As a class they are too poor to employ an attendant who charges anything like what it is worth to see them through their lying-in period. Practically none of them

are able t o have help in the home after the baby is born but depend upon a neighbor "running in" now and then to help what she can. To suggest a nurse is out of the question; to prescribe drugs, which are expensive and which practically NEVER have the least effect upon the milk production, is also unwise. The following set of directions is given to these women and they are instructed to call up daily and report to the office nurse their difficulties.

Take no medicine except a cathartic as needed.

Drink two quarts of water and two quarts of milk daily.

Eat any and all foods which agree with you.

Spend at least one hour daily out of doors.

Nurse the infant at ONE breast at the hours marked on your correction sheet and at NO OTHER TIME. STRIP THE BREAST AFTER EACH NURSING UNTIL DRY.

Lie down while nursing the baby. In this manner you will get more rest each day.

Take some form of recreation daily.

Follow these directions and you will have sufficient milk for your baby.

DO NOT WORRY. When in doubt, phone the nurse.

CASE HISTORY—RE-ESTABLISHMFNT

Mrs. R. W. R. Para I, family and personal history negative. Pregnancy uneventful and was delivered at a local hospital of a male infant weighing 6½ pounds. Her attendant was very watchful of the case and, because the infant weighed less than the usually considered normal weight, the infant was put to the breast every two hours. On the ninth day the mother and babe returned to their home and on the sixteenth day the mother resumed her household duties. The infant cried after each feeding, but was forced to the breast every two hours, upon the advice of the attending physician.

For six weeks he was unhappy, gained and lost and continued to vomit, and wh en seven weeks old was brought to me for advice. Breast stripping, proper feeding schedule and advice to the mother resulted in a satisfactory improvement as shown by Chart 2.

CHART 2—RE-ESTABLISHMENT OF BREAST MILK.

Date 1921	Age Weeks	Weight Pounds	Milk Secreted in ozs.	Totals Ounces
April 8 to April 16	7–8	6 9/16	Hours 6 9 12 3 6 10 2	
			By child .....0 .7 1 1 2 2 4	—10.7
			By hand .....1 .5 2 .9 1 2 3	—10.4
			Total in 24 hours, 21 1/10 ozs.	
May 22 to June 1	13–14	12 5/16	By child..3 3½ 3 2¾ 3 4 4	—23 4/16
			By hand..1 1¼ 1 .9 2 2 1¼	— 9 1/2
			Total in 24 hours, 32¾ ozs.	

In Group 3, we have mothers of practically every type. It has been my experience that the majority of women in this class are those who are active in public and social affairs. Many of them believe that if they have been able to supply breast milk for three months that from that time on the infant will thrive on most anything They frequently say, when questioned, that they had no idea that it was natural for breasts to function profusely for a longer period than three or four months. It has also been my experience that many of these women are keenly disappointed when a formula is refused them and they are instructed to continue nursing regularly and strip the breasts after each nursing. Frequently they tell me that they feel that they have "done their duty" by the infant when they stick to the routine of breast feeding for four months.

The wealthy mothers of this group are treated very curtly. It is pointed out to them that the only fair thing to do is to continue nursing and try to develop more milk. Those who will do this are given every possible support and aid; the others are instructed to go elsewhere for service.

The middle class mother of this

group is easy to handle. She has had experience with bottle feedings, usually, and actually knows that it is much easier and more satisfactory to feed from the breast. Often she hints that there is less likelihood of her becoming pregnant while nursing her infant. While this is certainly nothing more than a myth, I allow her what little satisfaction she gains from it and pass her along by agreeing with her. All that she really needs is to be taught how to strip her breasts and with proper encouragement she goes on nursing for five or six months longer without any difficulty.

The very poor mothers of this group are frequently less intelligent than one would expect. Their faculty of reasoning has been poorly developed and they rarely ever grasp the facts in favor of breast feeding over bottle feeding. If allowed, they would argue for hours in favor of some of the so-called baby foods which are marketed in tin cans and pasteboard boxes. While not hopeless, their case requires considerable patience, frequent visits by the nurse or myself, and eventually a few of them learn to follow blindly the directions given them without actually believing in what they have been taught to do. We feel, however, that this type is especially worth working with. About a year ago questionaires were sent to many of the doctors throughout the state with the hope that valuable data might be compiled. Very few answers have been returned but most of the replies have been to the affect that *breast fed infants unquestionably withstand infection and the hot weather better than do bottle fed babies.* This expression certainly is encouraging because once we make it clearly known that 100 per cent of women can nurse their babies there will be absolutely no excuse for the use of the now over-worked "patented foods".

To test the works out in Phoenix, I enlisted the co-operation of Dr. Charles B. Palmer. From January 1, 1921, to July 30, 1921, all infants delivered by him were the offsprings of mothers who were physically able to furnish abundant breast milk. The mothers were encouraged to nurse regularly and to strip the breast after each nursing. Fifty-two of these cases were followed for a period of six months. At the end of that time 97.6 per cent of these infants were still being fed from the breast EXCLUSIVELY. One case is especially interesting and briefly is reported as fololws:

Para 5, healthy, but had lost all previous children at birth. he came under the care of Dr. Palmer at the eighth month and a Caesarian seemed indicated. She was so delivered of a female infant weighing 5 pounds 14 ounces. The mother made an uneventful recovery and the breasts were stripped regularly after each nursing for four months, at which time the infant weighed 11 pounds 6 ounces and was able to practically empty the breast. I did not see the case again until the day before the child was one year old. On that date she weighed 19 pounds 4 ounces, being almost two pounds above weight for her age and sitting height.

What is true of a healthy mother in Minneapolis has been found equally true of the same type of woman in Phoenix. Because of the fact that in Arizona, Phoenix especially, we have very inadequate means of properly handling real sick children and also because it is quite generally believed that the very hot weather makes the production and delivery of milk which is perfectly safe for infants, almost impossible, we should all, without exception, gladly encourage more mothers to nurse their babies during the greater part of the first year.

Impressions gathered would almost lead one to presume that:

1. Doctors and nurses teach maternal nursing, but not so thoroughly as they should.

2. Mothers of all classes are eager for advice regarding the feeding o their babies. Are we all conscientious enough regarding the advice we give?

3. Stripping the breast will help every instance. Proper food, exercise, recreation and general right living should be encouraged as well.

4. There is no such thing as the milk of the mother biing "poinson" to her infant. It is far the best food to be had.

5. Knowing these facts, there is no excuse for the extravagant use of artificial foods.

BACTERIAL GROWTH IN THE GASTRO INTESTINAL TRACT: BIOLOGICAL NOTES*

ELLIOTT C. PRENTISS, M. S., M. D., El Paso, Texas.

In our contact with digestive diseases we are quickly impressed with the fact that bacterial infection plays a very important role. A study of bacterial growth in the gastro-intestinal tract should aid us in our grasp of both diagnosis and treatment, as it involves fundamental conditions. In nearly all cases of chronic digestive diseases, sooner or later, bacterial infection is one of the factors to be considered. It may not be directly related to the primary cause, but the results are such as to render secondary bacterial invasion almost certain. This can be readily understood when we remember that bacteria are constantly present on the mucous membrane of the whole digestive apparatus, and that practically all of these bacteria are capable, under conditions favorable to them, of producing or aggravating pathological conditions, but usually do not do so on account of the fact that normal tissue resistance prevents it.

The bacteria present are implanted in various ways, and remain because conditions are favorable to their growth; their existence represents the sum total of their ability to grow and the existing physical conditions in the gastro-intestinal tract.

For our purposes we might subdivide the bacteria found into four groups: 1. Those capable of producing specific diseases, as typhoid fever, cholera, dysentery, etc. 2. Those capable of producing severe infections, but not a definite specific disease, and not usually doing so, even when present, such as the pus cocci, streptococci, pneumonococcus, etc. 3. The predominating normal flora, which may under special conditions produce or aggravate lesions, such as the colon bacillus. 4. Those which are not normal inhabitants and are not known under any ordinary conditions to cause pathology. This is probably the largest group, and constitutes all the other bacteria ingested.

The above is intimately related to the subject of parasitism and infection, and the definition of these terms should be of interest. Theobald Smith (1) states that "In pathology it has been customary to distinguish between parasitic invasion and bacterial infection, the former producing relatively slight disturbances in the host, the latter the acute, often highly fatal, epidemic and epizootic diseases. The distinction is useful, but it can be made to apply only to extremes. Gradations of all shades occur. For the sake of more exact terminology, the distinction between invasion and infection might be made to hinge upon the capacity of the parasites to multiply in the host. It is thus best to class all living invasive organisms as parasites, subject more or less to the same host mechanism of repression and destruction. At one end of the scale are the highly specialized forms, adapted to one host species or even one race. At the other end are types merging from the predatory or saprophytic stage and acquiring parasitic habits."

The question of whether bacteria can grow in the digestive tract without causing some injury is interesting. I doubt if it is ever done. The injury may be very slight and remote, but it is probably there.

Metchnikoff states that the aging process is hurried by absorption of bacterial toxins or products of bacterial action on food from the intestines, even those produced by the normal flora, and intimating that this occurs even when motility is normal, and no pathological condition present. I believe that he is correct in this, and it fits in very well with what we know about life in general.

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In culture media bacteria at first grow very rapidly, then growth slows down and finally ceases, but the bacteria live for a variable period, and finally die. When charted this constitutes a definite life curve. The principal cause of this slowing down and final death is the toxins that have been thrown into the medium by the bacteria, and which are injurious to them. These toxins are not removed from the medium. A living organism cannot live indefinitely in its own excretions, no matter how favorable the other conditions may be. Neither can the human being do it. The removal of the poisons from the life medium is necessary, and failure to remove them, or the inability of the organism to destroy them, is injurious in proportion to their character and amount.

The constant absorption of bacterial toxins from the intestinal tract is bound to have its effect, however mild and remote. The production of liver cirrhosis, arteriosclerosis, and certain cases of severe anemia clearly indicate this. Bacterial toxemia of whatever origin may be taken as an example.

There are a number of factors that directly influence bacterial growth in the digestive tract. The temperature and moisture may be considered ideal. The maximum growth may occur at points a little above or below those present in the intestines, but the margin is only slight.

The various bacteria found have different cultural characteristics; that is, they prefer to grow upon certain substances under special conditions. The flora existing in any part of the digestive tract at a certain time are there because they find conditions favorable which other bacteria cannot survive. They are different in different parts of the digestive tract, as is shown by killing an animal and making cultures of the contents of the digestive tube in various segments from the stomach to the anus.

The small variety of bacteria found in the feces is due not only to factors unfavorable to their growth, but to the fact that those that grow best crowd out the others; a directly antagonistic effect against them. This no doubt varies from the more easy utilization of available food to direct biological antagonism.

The growing of bacteria on culture media is necessary for their isolation, identification and study, but in the digestive tract the medium is composite, as a mixture of foods is ingested. Owing to the preference of various bacteria for special media the character of the flora can, within certain limits, be changed at will. It is only necessary to mention here the vegetable and meat diet, the implantation of the lactic acid bacilli with buttermilk, and recent work with the bacillus acidophilus, especially in combination with lactose and dextrin. The contrast between the flora of intestinal putrefaction and fermentation is marked.

Gastro-intestinal motility is of great importance. While the number of bacteria is not directly proportional to the time of retention of food, the longer the delay the more time there is for the local irritation of the intestines, and the absorption of toxins.

There does not seem any good physiological reason why food should be retained in the colon as long as it is. Metchnikoff's work on this point is convincing. If we had only an eight-hour retention in the colon we would no doubt be better off, work harder and live longer.

When there is stasis it is practically always in the colon. X-ray examination demonstrates that there is seldom any barium in the ileum 24 hours after ingestion, no matter how marked the colonic stasis is.

The chemical reaction has an important influence upon the character and number of bacteria. The hydrochloric acid in the stomach no doubt exerts a strong inhibitory effect on the growth of the ordinary bacteria, especially those prevalent in the intestines, otherwise the colon bacillus would be a constant and thriving inhabitant of the stomach. Hydro-

chloric acid is not, however, the powerful antiseptic that many think it is. A recent article by Nicholas Kopeloff (2) shows that the number of bacteria in the stomach is largely dependent upon the amount of saliva swallowed and its bacterial content. We also have to bear in mind that there are many instances in which hydrochloric acid is subnormal in the stomach contents, or absent from it.

The period of retention of food in the stomach is long enough for the establishment of a characteristic and abundant flora, but this does not exist, owing to several factors, hydrochloric acid, and especially the gastric motility and the immunity of the gastric mucosa. But here we must remember that the colon bacillus does not thrive in the stomach even in the absence of hydrochloric acid.

The reaction in the small intestine is alkaline, unless changed by fermentation, in which case the acidity is the result of bacterial or yeast growth. Any change from the alkaline reaction, either in the small or large intestine, is due to such growth, or a too starchy or carbohydrate diet.

The secretions exert some effect on bacterial growth, but mostly indirectly. The hydrochloric acid of the stomach has just been mentioned. There does not seem to be any particular antiseptic property in pepsin, bile, pancreatic or intestinal juices, and if they aid bacterial growth other than by their reaction it is not known.

It is another matter, however, with the products of the action of these secretions on the food. Bacteria grow better, as a rule, on peptone and other products of proteid digestion than they do on meat, and the same is true of the sugar and other products of starch digestion in their relation to starch. Peptone and sugar resulting from ferment action are both found in the test-meals. The same is probably true of the products of fat digestion.

The gases found in the gastro-intestinal tube are probably of more importance than ordinarily believed. The gaseous medium in which all animals live is considered of vital importance, and the same applies to plant life. Bacteria, however, seem to have more leeway, especially with regard to oxygen. The nitrogen in the air swallowed with food, as far as we know, seems to be inert.

The gaseous products of bacterial growth, as mentioned above, unless removed, should tend to inhibit the further growth of the bacteria producing them. Its removal is accomplished by absorption of the gas and its expulsion by peristalsis. Here also we must consider the influence of such gases on the further growth of the other bacteria that did not produce them. This would, if other factors remained the same, usually be inhibitory.

The following gases may be mentioned as being present under varying conditions: Hydrogen, oxygen, nitrogen, marsh gas, carbon dioxide, sulphuretted hydrogen, and mercaptan.

What is the influence of these gases on the epithelium? With normal digestion there is probably none, but when gases are present in considerable amounts there would be more or less distention, with pressure, and there would be a larger proportion of gases known to be irritating, such as hydrogen sulphide. This could readily still further stimulate bacterial growth by lowering the natural epithelial resistance due to local irritation.

In studying the bacteria of the gastro-intestinal tract we implant the material on special media and reimplant, and thus isolate them and study details. We must remember that neither the media nor the conditions are the same as found in the intestinal tract. It would be interesting to conduct a series of experiments simulating intestinal conditions, except for the presence of intestinal tissue, using the various factors singly and in combination. The use of various articles of ordinary diet, singly and in mixture, varying the reaction, and with and without acid, pepsin,

bile, pancreatic juice and ferments, and the intestinal juices, and implanting the contents of the stomach and intestines as well as the isolated bacteria. With proper care the various gases found in the intestinal tract could be placed in the test-tubes, as well as mixtures of them. In ordinary work air is the gas medium used, but this does not represent the condition in the intestinal tract.

The large number of bacteria in the feces attest very strongly the favorable conditions for their growth in the intestinal tract. In normal feces they constitute about 25 to 30 per cent of the dried material. They must multiply with enormous rapidity to attain such numbers, as the average retention of food in the gastro-intestinal tract is only 36 hours. A point of interest here is the fact that most of the bacteria are dead when passed; only about 1 to 5 per cent are living, suggesting that there are powerful antiseptic factors at work constantly in the intestinal tract. This constitutes an apparent anomaly. On the one hand conditions encouraging extremely rapid bacterial growth, as we might say, in an almost unlimited manner, and on the other hand, and at the same place, an almost equally rapid killing of the bacteria.

In many instances of severe pathology in the intestinal tract I have had cultures made from the feces, but in practically none has it been of any assistance in either diagnosis or treatment, the report usually being a pure culture of the colon bacillus. Sometimes cocci grew, but very seldom the streptococcus. This was in spite of the fact that the stools were swarming with bacilli of varying sizes and shapes, and also innumerable cocci, some in fair-sized clumps. Infection of adults with the dysentery bacillus in this region must be rare, as some suspicious cases of it were examined and reported negative.

We might say that sooner or later all the bacteria with which we are in contact gain access to our gastro-intestinal tract. This applies equally well to our respiratory tract, yet the flora of the two regions are very different. What is the cause of this difference? We would naturally answer right away, "the food," yet the food element does not exist in the respiratory tract, and if an individual were placed on nothing but a boiled water diet for several days with thorough cleansing of the mouth, all the bacteria could not be washed out with salines, and the dejecta would still contain the characteristic flora. Restoration to food would then give the same findings as before the diet.

It seems evident that the tissues are the determining factor. The primary and necessary place of growth is evidently the intestinal epithelium, with the food playing an important secondary role. I believe that the culture experiments suggested above would demonstrate that. The colon bacillus predominates above all others probably on account of its greater affinity for the intestinal mucosa.

Innumerable other bacteria which gain access to the intestines do not thrive, no doubt, on account of the absence of this affinity.

We might here again refer to the stomach. The colon bacillus does not thrive there because the gastric epithelium must be strongly antagonistic to it. The acid cannot account for it, as it is frequently absent from the stomach contents without the colon bacillus being implanted.

I do not mean by the above that the bacteria do not grow in the food, as most of them found in the feces do multiply in that way, but I do mean that the determining factor in the nature of the flora is the specific influence of the epithelium.

Is there any damage done to the normal intestinal epithelium by the normal flora growing within normal limits? The answer must be "yes", but we do not know to what extent. Bacteria living on epithelium, that is, using it and the fluids exuded as food, must damage it in the process. This damage must be slight, but at present we have no way of estimating it. Toxins are produced by this nor-

mal flora, which, when found in large amounts in excessive bacterial growth, are known to be very injurious; so we may assume that they are also injurious, but to a smaller degree, in much smaller amounts. Under normal conditions, of course, the injury is slight and readily overcome. This local irritation is constantly going on, as is also the absorption of the poisons into the portal circulation. As mentioned in the early part of the paper, these toxins probably exert an inhibitory effect on the excessive overgrowth of bacteria in the intestinal tract.

The constant swallowing of bacteria from areas of focal infection frequently has an important influence on the bacterial content of the intestines. Instances of this are pyorrhea, chronic tonsillitis, gastric and duodenal ulcer, gall-bladder and liver infections, and local pathology in the intestinal mucosa. This can, and frequently does, initiate local infections, perhaps more than we know at present, and is always a source of danger when there is some primary area of pathology in the digestive tract. It has always seemed remarkable to me that the constant swallowing of tuberculous sputum, especially in the late cases, causes as little digestive trouble as it does.

In closing, I might say that this field is so large that a single paper can hardly do more than touch upon some of the interesting points, but can open the subject to a profitable discussion.

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(1) Science, New Series, Vol. LIV, No. 138; Aug. 5, 1921; p. 102.
(2) Nicholas Kopeloff, Jour. A. M. A., Feb. 11, 1922; Vol. 78, No. 6.

IMPORTANT ANNOUNCEMENT

Of more than usual interest to the medical profession is the announcement that the Abbott Laboratories, of Chicago, have purchased the Dermatological Research Laboratory, of Philadelphia.

The Abbott Laboratories is one of the leading manufacturers of "Council Passed" medicinals, and this addition to their products will give them added and well-deserved recognition from the medical profession everywhere.

It will be remembered that the Dermatological Research Laboratory was the first in the United States to produce arsphenamine during the war, when there was such a scarcity of this article, and this laboratory became well known to the profession for their patriotic attitude in developing and manufacturing medicinal preparations in this country. By the purchase of the "D R I" products, the Abbott Laboratories inherit their prestige.

The Dermatologic Research Institute, which became the property of the Abbott Laboratories on November 1st, will continue to operate in Philadelphia under the direction of Dr. George W. Raiziss, head of the Department of Chemistry, and his corps of specially trained assistants, who have been connected with the institute from its beginning. Orders for the "D R I" products will be promptly filled from the Philadelphia laboratory, from the home office of the Abbott Laboratories, Chicago, or from any of their branches or distributors.

For further information regarding their purchase of the Dermatologic Research Laboratory, the readers of this journal are referred to the statement of the Abbott Laboratories, on another page of this issue, entitled "Important Announcement to the Medical Profession."

EL PASO COUNTY MEDICAL SOCIETY

The El Paso County Medical Society met November 6 with 41 members and 5 visitors present.

Dr. R. B. Homan reported a case of a man, age 36, who swallowed a dental crown on June 17, 1922. Shortly atferward he became very ill with symptoms which led to a diagnosis of typhoid, but on development of cough and hoarseness, an x-ray was taken of the chest which showed the crown lodged in the right bronchus. This was successfully removed on July 20, but the cough did not let up entirely. The patient, who was at that time living in New York, went on an automobile tour and while in Buffalo had a large pulmonary hemorrhage on July 28. He immediately came to the southwest, and while on the way had to be taken from the train at Houston because of another large hemorrhage, but subsequently came to El Paso. At the present time he has a slight cough with no expectoration except that which comes from the nose and throat. Repeated examination of this material has been negative for tubercle bacilli. He has a slight elevation of temperature on exercising and since being here has had many small hemorrhages, but no large ones. Physical signs in the chest are practically nil. Dr. Homan thinks the patient is not tuberculous, but that the bleeding comes from an ulcer at the site of lodgement of the crown and he proposes to compress the right lung as treatment.

Dr. H. H. Stark reported and demonstrated a patient, a workman in one of the local railroad shops, who was struck some time ago in the right eye by a piece of steel. The body entered the eyeball, the wound of entrance being about five mm. up and out from the cornea. From the situation of the body on an x-ray plate it was thought that it had passed through the eye. It was localized with a Sweet localizer and found to be 6 mm. behind the orbit in the orbital fat. Both the wounds of entrance and exit have now healed and the scars could be easily seen with the ophthalmoscope, and there has been no interference with vision, it being entirely normal in the eye.

Dr. H. S. White reported a case of a man, age 56, who for the past ten years has been from pillar to post, visiting doctors, osteopaths, scientists, etc., for bladder trouble. He has been an invalid for some years and leading a catheter life. An x-ray, none having been previously made, showed three stones in the bladder, about one-half inch, one inch and two inches in diameter, respectively. The small stone was buried in the bladder neck and acted as an obstruction. Upon operation a small, contracted bladder was found which resembled a chicken gizzard in appearance. The stones were removed and the patient is rapidly recovering and is now able to retain a pint of urine, this being six months after operation.

Dr. H. S. White reported another case of a boy, age 17, an epileptic for three years, who gave a history of a hard instrumental birth, but no other history of trauma. The attacks at first were at intervals of from one to two weeks, but have gradually grown more frequent until during the past year they have come as often as three times a day. This boy had also received treatment from a large number of sources without relief. An x-ray of the skull showed a calcified area about one inch in diameter in the right parietal region about one-half inch below the surface. At operation enlarged veins were found radiating from the calcified area ,out over the surface of the brain. The area was easily found and, on opening, was found to be a small cavity lined with calcium salts. This was thoroughly cleaned out and the wound closed without drainage. For a month after the operation the patient continued to have "poundings in the head" similar to what he had experienced before the operation, but had no epileptic attacks. The so-called pounding gradually disappeared and now, four months after operation, there have been no further attacks and the patient seems entirely well.

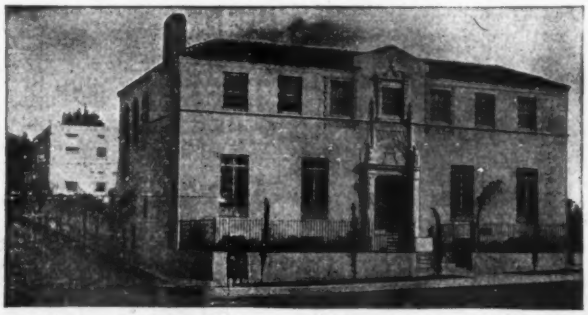
Dr. G. Werley demonstrated two small tubes of equal size and caliber, one containing 16 drops of Tr. Digitalis and the other containing 16 minums of the same tincture. The 16-minum tube seemed to contain about twice as much of the liquid. Dr. Werley gave this demonstration to show that the method of measuring by drops is very inaccurate and that when a patient is supplied with a bottle of Tr. Digitalis and told to take a certain number of drops, presumably an equal number of minims, he will not get the required dose of the drug, and consequently the desired action will not be obtained.

Drs. H. Crouse and W. S. Larrabee demonstrated four interesting x-ray plates. The first was that of a calcified pituitary gland in a woman, age 40, the mother of three children, who had been melancholic practically all her life and who a few years ago was confined to an insane hospital. On being told the nature of her trouble and having been shown on the plate the site of her trouble she apparently tried to shoot out the calcified area. Autopsy disclosed the pituitary gland as described. Her death prevented Dr. Crouse from trying massive doses of pituitary extract.

The second plate was that of a skull of a girl, age 8, who gave a history of epilepsy, but who was otherwise very bright. The picture showed marked syphilitic changes in the parietal bones and sella turcica.

The third plate was also of a skull of a man, age 23, showing syphilitic changes in the parietal, frontal, occipital bones and the sella turcica.

(*Continued on page* 446)



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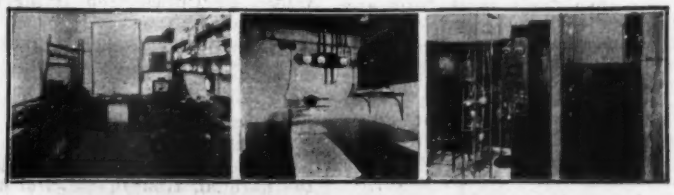
REX DUNCAN, M.D.
Medical Director

CALVIN B. WITTER, M.D.
Roentgenologist

EDWIN D. WARD, M.D.
Assistant Medical Director

T. C. CROWELL, M.D.
Pathologist



The fourth plate was that of an osteosarcoma of the knee in a man, age 24, which shows a tendency to disappear under x-ray treatment.

Dr. D. E. Smallhorst read a paper, "Foods and Feeding", which was discussed by Drs. Prentiss, Werley, E. A. Duncan, W. R. Smith, R. B. Homan and Major T. E. Scott.

Dr. M. I. Neff read a paper, "Some of My Experiences in Eastern Europe After the War", which was discussed by Drs. J. W. Tappan, H. H. Stark, and H. Crouse.

Dr. H. H. Stark reported all arrangements completed for the forthcoming annual meeting of the Medical and Surgical Association of the Southwest, which will be held at El Paso December 7, 8 and 9.

STAFF MEETING OF ST. JOSEPH'S HOSPITAL, PHOENIX

The regular monthly meeting of the open staff of the Sisters' Hospital, Phoenix, which was held on November 9th, was, by far, the best meeting so far held. Twenty members of the staff were present and engaged in a lively discussion of several cases recently treated in the hospital.

The first case discussed was one of cancer of the pancreas, with metastases to the liver. This was correctly diagnosed clinically and by the x-ray findings, which included pneumoperitoneum. The operation revealed conditions which could not be removed, and palliative cholecystotomy was done, as the common duct was involved in the malignant growth.

The second case was one in which all the findings pointed plainly to cancer of the colon, there being obstruction, cachexia, loss of weight, and x-ray filling defects in colon Operation failed to reveal the expected pathology. The causes for this error in diagnosis were discussed at some length.

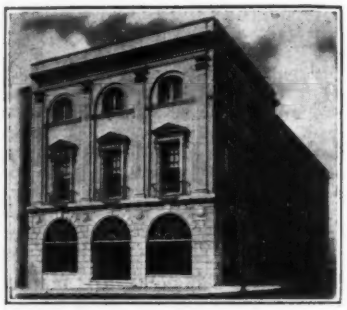
The third case was another error in diagnosis—the symptoms indicating acute appendicitis; following the removal of appendix, the pain continued and finally bowel obstruction forced a second operation, which showed massive adhesions of the small bowel, evidently of long standing, involving the entire small intestine—the entire mass being involved in a volvulus. This was relieved, but the patient developed pneumonia and died two days later.

Two cases, diagnosed acute nephritis, who died soon after entering the hospital, and another similar case just recently entering the hospital, were then discussed, the possibilities of error in diagnosis and the treatment administered being open for general criticism.

Radiograph of a case of dentigerous cyst was shown.

Two cases of Meniere's syndrome, or labyrinthine "stroke" were presented, with histories and the etiology and fine points in diagnosis discussed by the otologists present.

A resolution of condolence for the "poor dubs" who failed to attend the meeting was passed by unanimous vote.



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